

Myocardial infarction with intracoronary thrombus induced by anabolic steroids

Anabolik steroidlere baęlı gelişen intrakoronar trombus ile miyokard infarktüsü

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The use of anabolic steroids has been increased among strength trainers. Unfortunately, they are associated with adverse effects. We aimed to present such a case; an acute anterior myocardial infarction characterized by giant ST elevation, spontaneous resolution of coronary thrombus and distal embolization.

A 43 years old male patient was presented to emergency department with a squeezing in character chest pain of half-an-hour duration. Blood pressure was 120/70 mmHg and pulse rate was 55 beats/minute. With the detection of very high amplitude ST-elevation in leads V4-V6 (Fig. 1), 300 mg aspirin, 5 mg sublingual nitrate and 25 mg meperidine intravenously, were given and he was taken into catheterization laboratory for primary percutaneous intervention. Coronary arteriogram showed a very small plaque in the proximal left anterior descending (LAD) artery and occlusion in distal part of LAD and distal diagonal branch (Fig. 2) ST-elevation had been halved at that time. We concluded that LAD was occluded with a thrombus and distal coronary embolization had occurred. The patient was admitted to the coronary care unit. Clopidogrel was started (300 mg and 75 mg/daily thereafter) and tirofiban infusion was administered for 48 hours. T waves in anterior derivations had become negative on the second day. On echocardiographic examination ventricular walls were normal in thickness, but there was hypokinesia in apical septum and apex with an ejection fraction of 50 %. We found out that the patient was a strength trainer and was using drostandon proprionate intramuscularly, testosterone proprionate intramuscularly and methandrostenolone per os. Total cholesterol was 152 mg/dL, LDL cholesterol 123 mg/dL, HDL 14 mg/dL, triglyceride 73 mg/dL, CRP 0.3, anti-cardiolipin antibody Ig M 7.3 IU, Ig G 13.3 IU, homocystein 13.87 IU. Statin and angiotensin converting enzyme inhibitor were added to the treatment. After three weeks a treadmill exercise test was performed. He achieved 13 METs without chest pain and no ST-segment depression was associated.

Discussion

The use of anabolic-androgenic steroids has increased over the past decade. Athletes take them to enhance muscle mass

and physical performance, and those with a chronic wasting or malignant disorder take them to improve their physical appearance and strength. However, several cardiovascular complications including hypertension, cardiomegaly, stroke, pulmonary embolism, fatal and nonfatal arrhythmias and acute myocardial infarction are associated with the use of anabolic steroids (1).

The reported frequency of cardiovascular events among people taking anabolic steroids is probably underrepresented. It is often difficult to find competitive athletes for cardiovascular investigations who admit that they have taken anabolic steroids.

There are several reports of young competitive patients using anabolic steroids who experienced nonfatal myocardial infarction. Some had normal coronary arteries whereas others had occluded coronary arteries with thrombus on coronary angiogram (2-3). In a 12-year follow-up study risk of death was found to be 4.6 times higher among power lifters (4).

Anabolic steroids are potentially atherogenic through their actions on lipid metabolism. In a literature review, consistent and dramatic changes were observed in serum lipid levels of weight-lifters taking large doses of the hormones: overall, low-density lipoprotein levels increased by 36% and high-density lipoprotein levels decreased by 52%. These alterations convey the potential for accelerating coronary artery atherosclerosis and an increased risk of coronary heart disease is estimated to be 3-6 times of normal (5).

Possible mechanisms for an increased risk of arterial thrombosis due to anabolic steroids included increased levels of several procoagulant factors, decreased fibrinolytic activity, and increased platelet aggregation, decreased synthesis of prostacyclin. On the other hand androgens have been shown to increase heparin cofactor II, Hageman factor, protein C and protein S concentrations (6).

In the absence of any other possible risk factor for coronary thromboembolism the presented case that provides knowledge on growing association between anabolic steroid abuse and adverse cardiovascular events.

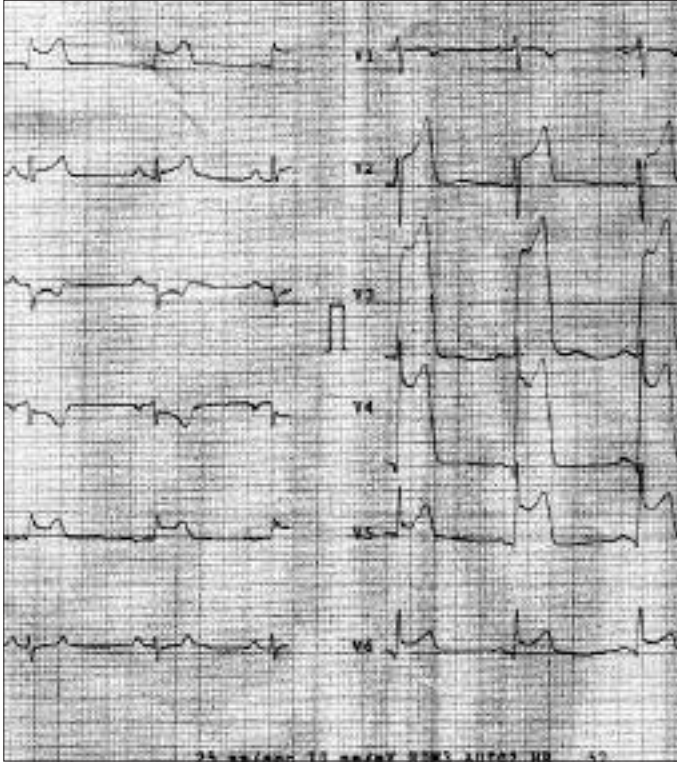


Figure 1. Electrocardiogram recording demonstrating acute anterior myocardial infarction with high amplitude ST-elevation

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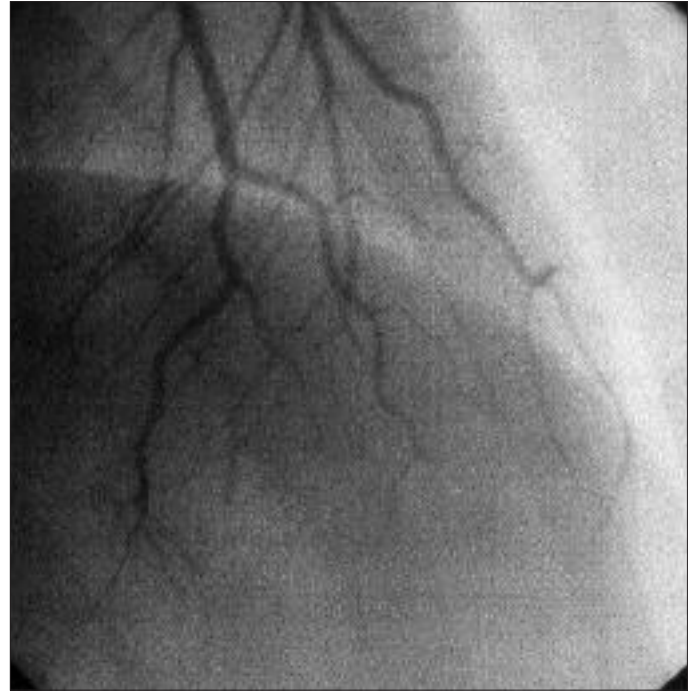


Figure 2. Coronary angiography demonstrating occluded distal left anterior descending artery and a side branch of diagonal artery.

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